

cases in compensating the valvular defect, at last give place to dilatation of the heart-cavities?

So far as the aortic valves are concerned, it would appear from the researches of Martin, Huchard, Weigert, Ziegler, and others that the change from a condition of hypertrophy to one of dilatation is coincident with involvement of the coronary arteries in the process of sclerosis and atheroma of the root of the aorta so constantly met with in aortic valvular disease. The blood-carrying power of the coronary arteries being thus impaired, there result changes in the cardiac muscle (increase of interstitial connective tissue and degeneration of the muscle-cells), in consequence of which its contractile power is lowered and dilatation ensues.

In disease of the mitral valve, however, the conditions are different, yet the most pronounced dilatation is often associated with that lesion. In the hope of finding some structural change in the heart-muscle which would suggest an explanation of this dilatation, BANTI (*Centralblatt f. Allgemeine Pathologie u. Pathologische Anatomie*, 1895, vi. Nos. 14, 15) has examined a number of hearts in various conditions of valvular disease, as the result of which he has been led to conclude that the conditions leading to cardiac dilatation in mitral disease are substantially the same as those which induce it in aortic lesions.

Hearts from three sets of cases were examined, representing different stages in the course of the disease. The first of these were patients in whom compensation was good, death having resulted from some intercurrent disease. In these, simple hypertrophy of the heart was found, without discoverable lesion in the muscular tissue.

The second class of cases comprised patients who were beginning to evince signs of failure of compensation in occasional attacks of cardiac failure, which were, however, as a rule, slight, and quickly responded to treatment. In these cases death resulted frequently from embolism or other complications. Here examination of the heart revealed great dilatation of the coronary veins, extending to their remotest ramifications within the heart-muscle. The intermuscular connective tissue appeared swollen, many of its cells being in a condition of parenchymatous degeneration. The muscle-cells were in places undergoing degeneration, as was shown by vacuolation of their protoplasm. In a few of these cases there was distinct increase in the connective tissue immediately surrounding the dilated veins, a kind of phlebo-sclerosis, which was usually accompanied by slight fatty degeneration of the muscle-cells in the immediate neighborhood.

In cases of more advanced cardiac disease, in which death was directly traceable to the condition of the heart, and in which dilatation was pronounced, the changes just described were found to be much more marked. Dilatation of the venous channels was excessive, their walls were greatly thickened, and there was great increase of the intermuscular connective tissue in their neighborhood, the newly formed connective tissue extending outward from them in all directions into the muscle-tissue. The muscle-cells could be seen in various stages of degeneration—atrophied, vacuolated, and more rarely fatty.

The distribution of these lesions in the heart-wall was not, as a rule, uniform. They were usually much more marked in the ventricular walls than

in the septum or auricles, and were by no means equally extensive in all cases.

The changes in the myocardium underlying dilatation and cardiac failure are, then, substantially alike in both aortic and mitral disease. In both they consist in connective-tissue growth and degeneration of the muscle-tissue consequent on impairment of the circulation in the heart-wall, in the one case resulting from disease of the coronary arteries, in the other from long-continued and excessive congestion of the coronary veins.

As accounting for the relatively early appearance of cardiac dilatation in mitral disease, Banti lays much stress upon the inherent weakness of the right side of the heart, which renders it less able to adjust itself to the increased strain thrown upon it than is the more powerful left ventricle.

THE PRESERVATION OF ORGANIZED URINARY SEDIMENTS.

It is often very desirable for purposes of instruction or demonstration and for comparison at different times in the course of a case of nephritis to be able to preserve any organized sediment which may be present in the urine.

This, according to FISCHER (*Prager medicinische Wochenschrift*, 1895, No. 12), may be accomplished by washing the sediment two or three times with normal salt-solution, best with the aid of the centrifugal machine, and preserving it in equal parts of glycerin and water to which a little (about 2 per cent.) saturated alcoholic solution of thymol has been added.

Sediments treated in this way preserve their structural characters indefinitely and may at any time be mounted for study with the microscope by simply placing a drop of the mixture on a slide, covering it, and preventing evaporation by a ring of varnish.

THE ELIMINATION OF URIC ACID AND XANTHIN-DERIVATIVES BY THE ALIMENTARY CANAL.

So much stress having lately been laid upon increase of uric acid in the urine as an indication of various disorders of nutrition, it is of interest to know that the occurrence of uric acid and certain xanthin-bases as normal excretions of the alimentary mucous membrane has been shown by WEINTRAUD (*Centralblatt für innere Medizin*, 1895, No. 18).

In a case of leukæmia in which there was a very great increase in the leucocytes, but only slight increase in the uric-acid excretion by the urine, a considerable amount of substance responding to the tests for the xanthin-group of bodies was discovered in the feces, amounting in terms of hypoxanthin to about one gram for the twenty-four hours.

On investigation a variable quantity of these bodies was found to be a constant ingredient of the feces. Their presence was, furthermore, determined to be quite independent of the character of the food. They were still present during a strictly milk-diet, although the decomposition of the nucleo-albumins of milk does not yield any of the-xanthin-group; and also when a non-nitrogenous diet was taken. They were detected in the accumulated meconium in a case of *atresia ani*. Nor was their quantity greatly increased by a diet rich in nucleo-albumins, as was shown by examinations made after the ingestion of considerable quantities of calf's thymus.